Anesthetic Management of an Incompletely Controlled Hyperthyroid Patient for Thyroidectomy

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Modern therapy with antithyroid drugs is so effective that the anesthesiologist is seldom confronted with an uncontrolled thyrotoxic patient. Although thyroid storm rarely occurs in well controlled patients, it is likely to occur in poorly controlled patients.¹ Many of its manifestations resemble those of adrenergic stimulation: tremor, anxiety, tachycardia, arrhythmias, increased cardiac output, increased metabolic rate and increased temperature. Interaction of at least two, and possibly three, hormonal systems is necessary to produce these manifestations.² If either the adrenergic or the thyroid system is blocked, the hemodynamic and metabolic signs and symptoms of thyrotoxicosis can be eliminated or reduced. Spinal anesthesia,³-⁵ reserpine,⁶ guanethidine,⁶,⁷ and ganglionic blockade³ have been suggested to interrupt the adrenergic system and thereby control the peripheral manifestations of hyperthyroidism or thyroid storm. Sympatholytic drugs may alleviate the tachycardia, arrhythmias, tremor, restlessness and heat intolerance in hyperthyroidism. However, thyroid function itself is unaffected, with sustained increases in protein-bound iodine and radioactive iodine uptake.⁸ Cardiac output and oxygen consumption are decreased somewhat by sympathetic blockade but no change in weight or serum cholesterol occurs.

We recently were involved in the anesthetic management of a patient allergic to both methimazole and propylthiouracil who required thyroidectomy at a time when she was hyperthyroid. We used alpha- and beta-blocking drugs to prevent thyroid storm. The patients course and our rationale for this management are presented.

REPORT OF A CASE

A 14-year-old Caucasian girl known to have had hyperthyroidism for seven months developed a generalized pruritic erythematous rash, first in response to methimazole and then in response to propylthiouracil, in the course of her therapy. These drugs were discontinued and she was evaluated at Yale-New Haven Hospital on February 3, 1970. Symptoms included easy fatigability, tremulousness, emotional difficulties, heat intolerance and weight loss. The patient denied diarrhea or menstrual irregularities.

Physical examination showed a thin, anxious, girl with tachycardia of 132 beats/min. She had bilateral exophthalmos with bilateral lid lag. The thyroid was diffusely enlarged, soft, and nontender, with bilateral bruits. Deep tendon reflexes in the lower extremities were hyperactive. Serum protein-bound iodine (PBI) was 19.0 μg/100 ml;¹ with a T₃ uptake of 70 per cent.⁶

¹ Normal value: 4.0–8.0 μg/100 ml.
⁶ Normal value: 93 per cent uptake or less is compatible with hyperthyroidism.
On February 3 propranolol, 10 mg three times a day, and phenobarbital, 30 mg four times a day, were started. On February 17 Lugol's solution was added to the medication. On February 26 the patient was admitted for thyroidectomy, scheduled for March 2. Physical examination disclosed no changes from earlier in the month. We first saw the patient on February 27. On February 28 we increased her phenobarbital to 60 mg every fourth hour, and she was given phenolamine (Regitine), 50 mg orally, four times a day.

Anesthetic Management. On March 5 the patient was sedated with 200 mg secobarbital at 6 AM. Blood pressure was 125/80 mm Hg; pulse rate, 60 beats/min. At 8 AM she was given an addition 100 mg secobarbital and 5 mg phenolamine intramuscularly. Blood pressure was 140/80 mm Hg, pulse rate, 90 beats/min at 10 AM prior to patient's transfer to the operating suite. On arrival in the operating suite she was alert and anxious. Blood pressure was 120/80 mm Hg, pulse rate, 118 beats/min.

Thiopental, 50 mg, was administered intravenously as a test dose, without adverse effect. Anesthesia was then induced with 200 mg thiopental and inhalation of nitrous oxide-oxygen-methoxyflurane begun. Blood pressure following induction was 130/80 mm Hg, pulse rate, 90 beats/min. After 15 minutes of methoxyflurane anesthesia and preoxygenation with 100 per cent oxygen for three minutes, succinylcholine, 100 mg, was administered intravenously. Tetracaine, 30 mg, was applied topically to the vocal cords and trachea in divided doses; the trachea was then intubated with a #36 cuffed endotracheal tube with ease. The blood pressure rose from 110/70 to 140/80 mm Hg and the pulse rate from 90 to 108 beats/min concomitant with intubation. Maintenance anesthesia consisted of a mixture of 60 per cent nitrous oxide and 40 per cent oxygen, with 0.5 to 0.25 per cent methoxyflurane. Ventilation initially was controlled and then was assisted throughout the operative procedure. The blood pressure ranged from 120/60 to 140/90 mm Hg, the pulse rate from 80 to 110 beats/min. The temperature varied between 98.4 and 97.8 F. A subtotal thyroidectomy was performed in three hours. The patient was extubated at the end of the procedure. No attempt was made to have her completely awake. She was transferred to the recovery room in satisfactory condition.

On admission to the recovery room the patient responded to pain but not to verbal commands. Blood pressure was 140/110 mm Hg, pulse rate, 84 beats/min, temperature 97.8 F. An hour after reaching the recovery room the patient developed respiratory stridor associated with thick secretions. Direct laryngoscopy showed both cords moving well. The patient was discharged to her room at 10:15 AM in good condition, but with persistent large amounts of thick pharyngeal secretions.

On the first postoperative day the patient developed positive Chvostek's and Trousseau's signs, without tetany. She was given 3 gm calcium chloride diluted in 5 per cent dextrose in water intravenously, with gradual remission of Chvostek's and Trousseau's signs. She was discharged in satisfactory condition on the seventh postoperative day.

Discussion

Since thyroid storm resembles a sympathetic crisis, and since our patient was already taking propranolol, we completed her adrenergic blockade with phentolamine for the following reasons: 1) Beta blockade blocks inotropic and chronotropic responses to catecholamines, leaving intact the peripheral alpha constrictor response.11,12 Since our patient was to be exposed to the additional negative inotropicism of methoxyflurane, beta blockade alone conceivably could have led to myocardial failure in the presence of an intact or overactive vasoconstrictor mechanism due to uninhibited alpha activity.13 2) Beta blockade alone also leaves unopposed constrictor receptors in the bronchi which could precipitate bronchospasm. 3) Both systems must be blocked to prevent the peripheral manifestations of thyroid crisis. Phentolamine was chosen as a competitive alpha blocker for its relatively rapid onset of action, specificity and controllability.

Anticholinergics were excluded from the preoperative medication of our patient to avoid a possible increase in pulse rate or elevation of temperature in a hyperthyroid patient. Atropine administration has been reported as a test for the adequacy of control of hyperthyroidism.14 Barbiturates were used in premedication for their hypnotic and possible antithyroid effects.15 Sodium thiopental provided pleasant, rapid, induction and possible antithyroid action.

Supplementation of methoxyflurane with nitrous oxide-oxygen was chosen because this technique appears to elicit no thyroid activity, which is not true of ether or halothane.16 Methoxyflurane does not appear to sensitize the myocardium to catecholamines, does not appear to enhance vagal tone17,18 and provides excellent postoperative analgesia, which we felt was important. Finally, methoxyflurane is not a sialagogue. Since our patient did not receive anticholinergics preoperatively we wanted to avoid anesthetics that might stimulate secretions.
In the immediate postoperative period the patient had excess secretions. Since no anticholinergics were given, adrenergic blockade producing relative parasympathetic stimulation and the surgical dissection around the trachea probably contributed to secretion formation. At no time preoperatively or postoperatively did the patient have symptoms of postural hypotension.

REFERENCES


Drugs

ANESTHETICS AND WATER STRUCTURE Pauling and Miller proposed independently that the presence of an anesthetic gas in tissue induced a cage-like arrangement of hydrogen-bonded water molecules. If during anesthesia the number of hydrogen-bonded water molecules in tissue is increased, then the movement of an average water molecule should be hindered. A change in the flux of tritiated water could not be detected in rat cecum exposed in vitro to 20 per cent cyclopropane. However, the histologic injury in this tissue proved to be greater than those in identical tissues not exposed to cyclopropane. When the cyclopropane concentration was reduced to approximately 10 per cent, the histologic appearances were the same in exposed and unexposed tissue, and under these circumstances water flux in exposed tissue was delayed by 12 per cent compared with unexposed tissues. If the mechanism of this decrement is the same as predicted by theory, the present observations indicate that cyclopropane may induce minute hydrate crystals or an "ice cover" in tissues. (Berger, E. Y., Pecikyan, F. R., and Kanzaki, C.: Anesthetic Gas and Water Structure: Cyclopropane Effect on Water—H1 Flux across the Gut, Amer. J. Physiol. 217: 414 (Aug.) 1969.)